of gelatine plates and Mg and Ca salts, at least under certain conditions, retard the swelling and may even cause shrinkage. OH′ increases swelling and H· decreases swelling if the reaction of the medium is on the alkaline side of the isoelectric point for the colloid, which seems to be the case in regard to blood and animal proteins within the body or in sea water.

We may therefore conclude that OH′, Na˙, and K˙ increase the permeability of the plasma membrane by causing it to swell and that Ca˙˙, Mg˙˙, and H˙ (at least on the alkaline side of the isoelectric point) inhibit increase in permeability by inhibiting swelling.

[1] *J. Biol. Chem.*, 25, 669 (1916).

[2] These vials were obtained from Hynson, Westcott and Company, Baltimore, Maryland.

[3] There is reason to support the view that the cell surface is composed of emulsoids rather than suspensoids.

SOME INTERRELATIONS BETWEEN DIET, GROWTH, AND THE CHEMICAL COMPOSITION OF THE BODY

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Changes which normally occur in the water, ether extract, and ash content of the body during its most active growth have been determined for the white mouse. Based on eighty-eight analyses of the entire body at different stages of growth, the following results were obtained: (*a*) increase in solids from 16% at birth to a maximum of 35% at fifty days with a subsequent decrease to 33%; (*b*) decrease in the proportion of water in the fat-free substance from 85.5% at birth to 73% in the adult mouse; (*c*) rapid increase in fat during the first twelve days from 1.85% to about 10%, followed by a slow increase to about 12%; (*d*) absolute and relative increase in ash content from 25 mg., 1.86%, in a mouse weighing one and a half grams at birth, to 950 mg., 3%, of ash in the adult mouse.

The ash content of mice growing normally on an artificial food mixture of milk powder, casein, starch, salts, and butter fat, (protein 30%, fat 32%, and ash 5.5%), is uniformly higher at corresponding stages of growth than the ash content of mice fed on a ration of mixed grain, dog bread, and small amounts of milk. It is not known to what this difference is due, but it is not related to differences in the amount of protein or salts in the food. Since the ash content of normal animals may be thus increased by differences in diet alone, it would seem that

conclusions as to the development of the skeleton during arrested growth which are based on an increased ash content of the body, should depend upon comparison not with the 'normal animal,' but with the normal animal on the same diet.

When abundance of fat is furnished in the diet, but not enough protein to maintain normal growth, the fat content of the animal is greater than when the food contains an adequate amount of protein with the same proportion of fat. There seems to be a tendency to protect the limited amount of protein by increasing the available supply of fat in the body. This does not occur when growth is arrested by lack of lysine, as in the use of gliadin as the only protein in the diet, since in this case the limiting factor lies not in the amount but in the nature of the protein.

By underfeeding, mice have been completely arrested in growth, as far as growth is expressed in gain in weight, and have been maintained at a constant body weight of 12 grams for thirty and sixty days. At the end of the thirty-day period, control mice of the same initial weight as the experimental animals, and on the same diet, weigh 22 grams. Comparison of the composition of the stunted mice with that of mice growing normally shows that the proportion of fat in the stunted animal is about the same as in the normal mouse of the same weight (but younger), while the percentage of water in the fat-free substance corresponds to the water content of a normal mouse of the same age (but heavier). That there is no evidence of a general replacement of fat by water, such as is often reported in underfed animals, may be due to the large proportion of fat used in this diet. The ash content, both absolute and relative, of the stunted mouse is greater than that of the normal mouse of the same weight, confirming in this the results reported by Aron for the rat, and indicating continued growth of the skeleton under conditions which prevent the animal from gaining in weight. The tendency of the skeleton to develop under such adverse conditions does not appear to be as strong in the mouse, however, as in the rat, if the few data reported for the rat are representative.

Retardation of growth by other means—reduction of protein or of salts in the food, or substitution of gliadin for other proteins—affects the ash content of the animal in the same way as simple underfeeding.

If young mice which have been maintained for a time at constant weight are given sufficient food again, they grow at a greatly accelerated rate which enables them to overtake control mice which have grown uninterruptedly. The ash content, however, does not increase at the same rate as the body weight; and the development of the skele-

ton, as represented by changes in the total ash of the body, which proceeded for a time at the expense of other tissues while the animal was held at constant weight, does not now, in the active growth which accompanies refeeding, keep pace with this rapid gain in body weight, and consequently in a few days the normal relation is almost reestablished.

FURTHER STUDY OF THE ATOMIC WEIGHT OF LEAD OF RADIOACTIVE ORIGIN

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The recent independent and almost simultaneous investigations upon the atomic weight of lead from radioactive minerals have proved with very little room for doubt that the substance derived from this source has a much lower atomic weight than ordinary lead.[1] This conclusion is so important in its theoretical relations that its every aspect should be carefully investigated. Accordingly, the present paper represents further research in this direction, embodying determinations of the atomic weight of new samples of varied origin. The outcome entirely supports the earlier conclusion.

Four samples from widely separated sources were studied in the present research, namely, lead from Australian carnotite, from American carnotite, from Norwegian cleveite, and Norwegian bröggerite.

The first of these samples was obtained in large quantity through the kindness of Mr. S. Radcliff and Mr. E. R. Bubb, of New South Wales.

The preliminary purification of the sample was carried out in Australia.[2] Our subsequent purification was briefly described in a recent paper,[3] but some additions to the account are needed. The metallic lead was dissolved in nitric acid, leaving practically no residue. A portion of the nitrate thus obtained was precipitated with 20% hydrochloric acid from dilute solution. Lead sulphide was precipitated from the warm acidified solution of this chloride by pure hydrogen sulphide, and after separation and washing was dissolved in pure nitric acid. The small portion oxidized to sulphate during this process was boiled with sodium carbonate and the lead carbonate, washed free from sodium, was dissolved in nitric acid and united to the main portion of the nitrate, which was recrystallized four times from pure water and precipitated as chloride from a warm solution in a quartz